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**Follistatin interacts with Noggin in the development of the axial skeleton.**

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**Authors:** David A Stafford, Stefanie D Monica, Richard M Harland

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**Public Summary:**

Somites are transient embryologic structures that differentiate into precursors for skeletal muscles as well as bone and cartilage of the axial skeleton. Embryological and genetic analyses have identified the molecular signals that initiate somite pattern formation. Among these signals are members of the BMP family, as well as BMP antagonists that restrict their activity in specific cells of the developing embryo. In this and a previous study, we investigated the role of BMP antagonists Noggin, Follistatin and Gremlin in patterning the axial skeleton. We present evidence that Follistatin, Noggin and Gremlin1 participate in a "BMP antagonist relay" in early sclerotome development, consistent with a persistent function for BMP/BMP antagonist signaling during the differentiation of somite derivatives.

**Scientific Abstract:**

When compared to single mutants for Follistatin or Noggin, we find that double mutants display a dramatic further reduction in trunk cartilage formation, particularly in the vertebral bodies and proximal ribs. Consistent with these observations, expression of the early sclerotome markers Pax1 and Uncx is diminished in Noggin;Follistatin compound mutants. In contrast, Sim1 expression expands medially in double mutants. As the onset of Follistatin expression coincides with sclerotome specification, we argue that the effect of Follistatin occurs after sclerotome induction. We hypothesize that Follistatin aids in maintaining proper somite size, and consequently sclerotome progenitor numbers, by preventing paraxial mesoderm from adopting an intermediate/lateral plate mesodermal fate in the Noggin-deficient state.

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